Contraction of the human diaphragm during rapid postural adjustments

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1. The response of the diaphragm to the postural perturbation produced by rapid flexion of the shoulder to a visual stimulus was evaluated in standing subjects. Gastric, oesophageal and transdiaphragmatic pressures were measured together with intramuscular and oesophageal recordings of electromyographic activity (EMG) in the diaphragm. To assess the mechanics of contraction of the diaphragm, dynamic changes in the length of the diaphragm were measured with ultrasonography.

2. With rapid flexion of the shoulder in response to a visual stimulus, EMG activity in the costal and crural diaphragm occurred about 20 ms prior to the onset of deltoid EMG. This anticipatory contraction occurred irrespective of the phase of respiration in which arm movement began. The onset of diaphragm EMG coincided with that of transversus abdominis.

3. Gastric and transdiaphragmatic pressures increased in association with the rapid arm flexion by 13·8 ± 1·9 (mean ± S.E.M.) and 13·5 ± 1·8 cmH_2O, respectively. The increases occurred 49 ± 4 ms after the onset of diaphragm EMG, but preceded the onset of movement of the limb by 63 ± 7 ms.

4. Ultrasonographic measurements revealed that the costal diaphragm shortened and then lengthened progressively during the increase in transdiaphragmatic pressure.

5. This study provides definitive evidence that the human diaphragm is involved in the control of postural stability during sudden voluntary movement of the limbs.

The diaphragm is the primary muscle of inspiration. The abdominal and thoracic cavities on which this muscle acts are also involved in the stability of the trunk and postural control (Grillner, Nilsson & Thorstensson, 1978; Cresswell, Oddsson & Thorstensson, 1994). Other respiratory muscles acting on the ribcage and abdomen are known to perform a postural function that is integrated with their respiratory role. For example, activation of human intercostal muscles moves and stabilizes the ribcage. This activity is modulated by breathing (Rimmer, Ford & Whitelaw, 1995). Contraction of the abdominal muscles contributes to trunk stability prior to and during movement of the limbs (Hodges & Richardson, 1997*a*, *b*) and this action is increased when respiratory demands increase (Hodges, Gandevia & Richardson, 1997). In addition, studies of decerebrate animals provide evidence that intercostal muscle activity changes in association with stimulation of tonic cervico-labyrinthine reflexes (Massion, Meulders & Colle, 1960; Delhez, 1968). Although the diaphragm contracts in non-respiratory movements such as the expulsive effort of defecation and parturition (Agostoni & Sant'Ambrogio, 1970), the general view is that the diaphragm does not perform a postural function (e.g. Monteau & Hilaire, 1991).

The diaphragm may contribute to postural stability by increasing pressure within the abdominal cavity, thereby maintaining the hoop-like geometry of the abdominal muscles (Farfan, 1973; McGill & Norman, 1987) or by hydraulically unloading the spine and increasing trunk stability (Bartelink, 1957; Grillner *et al.* 1978; Cresswell *et al.* 1994). Contraction of the pelvic floor and abdominal muscles, particularly transversus abdominis, correlates closely with increases in intra-abdominal pressure in a variety of postural tasks (Grillner *et al.* 1978; Hemborg, Moritz & Löwing, 1985; Cresswell *et al.* 1994). When the stability of the trunk is challenged by reactive forces due to limb movement, transversus abdominis contracts *prior to* the agonist limb muscle and this suggests that the response may be preprogrammed by the central nervous system (CNS; Hodges & Richardson, 1997*a*, *b*).

For the contraction of abdominal muscles to elevate intra-abdominal pressure it is necessary for the diaphragm to

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contract simultaneously and thus minimize displacement of the abdominal contents into the thorax. The present study was designed to determine whether contraction of the diaphragm occurred as a component of the preparatory postural responses associated with rapid movement of the upper limb. In addition, we examined whether abdominal pressure increased prior to the initiation of movement of the limb and whether this was associated with shortening of the diaphragm.

METHODS

Five subjects participated in the study (4 male, 1 female, 25–44 years of age). Subjects were excluded if they had any significant respiratory or neurological condition. All procedures were approved by the institutional ethics committee and informed written consent was obtained.

Standing subjects were required to flex the left arm rapidly at the shoulder in response to a visual stimulus. In the main study the onset of electromyographic activity (EMG) in the diaphragm was recorded with intramuscular electrodes in five subjects and in transversus abdominis in two subjects. In all subjects the transdiaphragmatic (P_{di}), oesophageal (P_{oes}) and gastric (P_{ga}) pressures were recorded (Fig. 1*A*). Two additional investigations were performed. In the first, the EMG response of the diaphragm to movements of the thumb, wrist and elbow was measured as well as to movement at the shoulder. The second study involved ultrasonographic measurement of the length and change of length of the zone of apposition of the right hemidiaphragm during limb movements.

Electromyography and pressure studies

To record EMG from the diaphragm, Teflon-coated monopolar needle electrodes with a receptive area of 0·15 mm^2 were inserted into the costal portion of the right hemidiaphragm in the seventh or eighth intercostal space near the mid-clavicular line (Fig. 1*B*) after injection of 0·5–1 ml of lignocaine (lidocaine; 2% with adrenaline) superficially into the intercostal space (De Troyer, Leeper, McKenzie & Gandevia, 1997). The site was chosen based on an ultrasonographic examination of the intercostal space using a range of linear transducers (Acuson, XP128, CA, USA) and was close to the costal insertion of the diaphragm and below the reflection of the visceral pleura. Ultrasound measurements and the insertion of the electrode were performed with the subject standing. The tip of the electrode was confirmed to be in the costal diaphragm by the detection of phasic multi-unit EMG during inspiration but not with expiratory manoeuvres which activate the internal intercostal muscle. Repositioning of the needle was occasionally required between limb movements. The reference electrode was a surface disc placed over an adjacent rib 1–2 cm from the needle.

Transversus abdominis EMG was recorded on the right side with a pair of fine-wire electrodes about 2 cm anterior to the proximal end of a line running perpendicularly from the anterior superior iliac spine to the distal edge of the ribcage. The wires (75 mm diameter, Teflon-coated stainless steel) were twisted together and heated to fuse the insulation between them, inserted into a short-bevelled needle (0·70 mm × 38 mm) and bent back 1·5 mm at the end. The distal end of the wires was cut leaving only their tips exposed. Prior to insertion, the depth of the inner border of transversus abdominis was identified by ultrasound imaging. There were no adverse effects from any of the procedures.

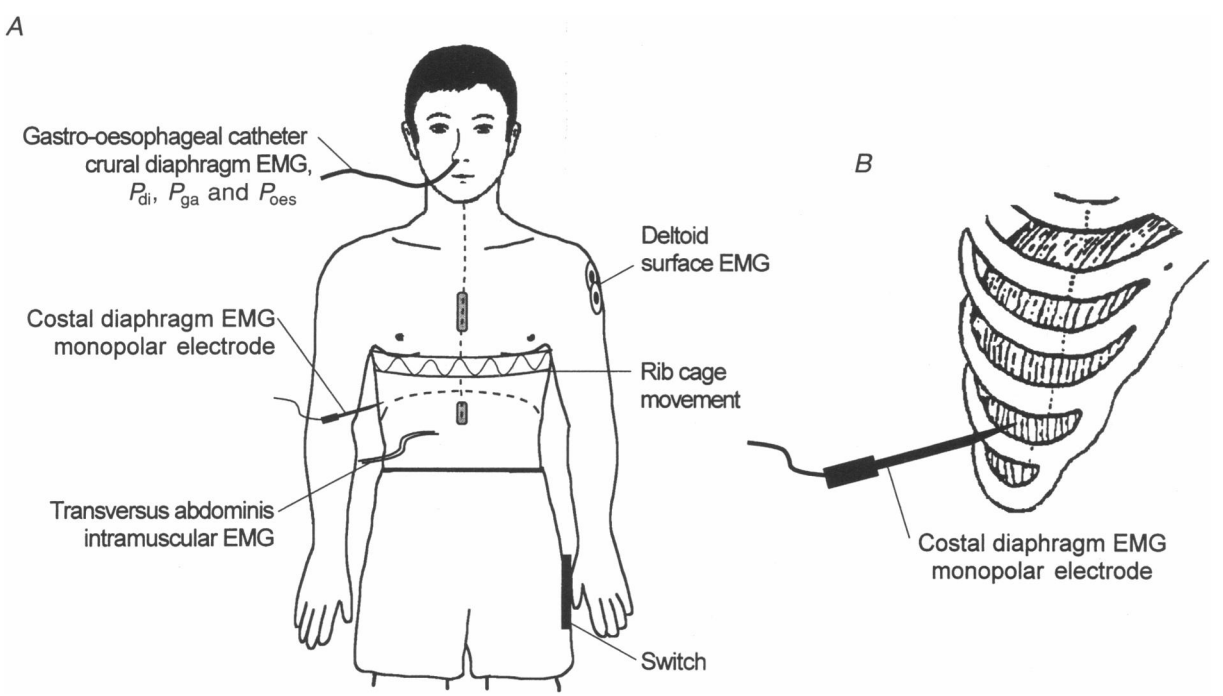

Figure 1. Methods

A, experiment arrangement. Transdiaphragmatic pressure (P_{di}), gastric pressure (P_{ga}) and oesophageal pressure (P_{oes}). *B*, diagrammatic view of the right anterolateral chest wall to show the site of needle insertion into the diaphragm.

A pair of surface EMG electrodes was placed over the belly of the left deltoid in parallel with the muscle fibres. In control studies EMG of deltoid was recorded with both surface and intramuscular electrodes. No difference in time of onset of EMG was detected between the two recording methods. The subject was earthed via a large flexible electrode placed on the left shoulder.

Oesophageal recordings of EMG from the crural diaphragm and the oesophageal, gastric and transdiaphragmatic pressures were made using a multilumen gastro-oesophageal catheter (McKenzie & Gandevia, 1985). Crural diaphragm EMG was recorded from two chlorided silver circumferential bands around the catheter (inter-electrode distance, 10 cm). The catheter was passed through the nose until the gastric balloon was in the stomach. The tube was positioned optimally and then fixed to the nose. The proximal balloon in the oesophagus (inflated with 0·8 ml of air) measured the mid-thoracic intrapleural pressure and the gastric balloon (inflated with 1·5 ml of air) measured the gastric pressure. The difference between these two pressures was an index of the pressure generated by the diaphragm (transdiaphragmatic pressure). Movement of the chest wall and thus respiratory phase was measured using an inductance plethysmograph placed around the chest at nipple level.

Evaluation of movements of the whole arm

The subject adopted a relaxed standing position and maintained normal respiration throughout the test period. Once the diaphragm electrode was positioned, flexion of the left shoulder was performed 'as fast as possible' to the horizontal in response to a visual stimulus to move. The stimulus to move occurred at a random point during the respiratory cycle. To ensure the reaction time was as rapid as possible, the subject was given an audible warning at a random period (0·5–2 s) prior to the stimulus to move. The initiation of movement of the arm was measured using a switch attached to the left thigh and activated as the arm moved away from the leg. The activity of the costal diaphragm during tidal breathing was monitored continuously by one of the experimenters. No auditory or visual feedback of EMG was available to the subject. Twenty repetitions of shoulder flexion were performed by each subject. In one subject, twenty consecutive recordings were achieved from a single costal diaphragm site that was characterized by the phasic discharge of a distinctive large single motor unit. The observations at that site were qualitatively similar to those at sites where multiunit activity was recorded. Due to the electromechanical delay the main measurement of the study, the time of onset of diaphragmatic EMG, was not affected by any contraction-induced movement of the diaphragm and/or needle. Trials were rejected on-line if the subject moved before the visual stimulus or failed to respond to it within 400 ms. Fewer than 5% of trials were rejected. In separate trials the duration of movement of the arm from thigh to horizontal was recorded using two switches.

Evaluation of movements of distal limb segments

In an additional study, a similar protocol was performed in two subjects who participated in the main study. The protocol was the same as above, including the instruction to respond as fast as possible, except that the standing subject was required, in different sequences of trials: (i) to move the whole arm to the horizontal (i.e. control task), (ii) to flex the elbow, (iii) to extend the wrist and fingers or, (iv) to abduct the thumb. For all conditions, the arm was initially resting vertically with the hand touching the thigh. This experiment was designed to determine whether an anticipatory response in the diaphragm would occur with movements produced by movement of limb segments of different mass. For this study intramuscular EMG from the costal diaphragm and surface EMG from the thenar muscles, extensor muscles of the forearm and elbow flexors were recorded. Subjects performed ten consecutive repetitions of rapid thumb flexion, rapid wrist and finger extension, and rapid elbow flexion in response to a visual stimulus presented at random times in the respiratory cycle.

Measurement of diaphragm motion

The length of the zone of apposition of the diaphragm to the chest wall correlates closely with the total length of the diaphragm (Petroll, Knight & Rochester, 1990; McKenzie, Gandevia, Gorman & Southon, 1994) and can be measured accurately with ultrasound (McKenzie *et al.* 1994). Ultrasonography was used here to measure dynamic changes in the length of the zone of apposition of the diaphragm during rapid arm movement in four subjects who participated in the main study. Measurements were made with a 12 cm linear array probe (3·5 MHz) placed longitudinally between the right anterior and mid-axillary line and held firmly against the chest wall to maintain the placement during movement of the arm. A switch was attached to the left thigh to indicate the onset of movement of the left arm and its output was recorded simultaneously overlying the ultrasound images. Images were sampled at 28 frames s^{-1}. The procedure was almost identical to the main study. Subjects performed five to ten repetitions of rapid shoulder flexion in response to an auditory stimulus to move. All repetitions were performed at the resting end-expiratory volume. The costal origin of the diaphragm and the proximal extent of the zone of apposition were identified during inhalation to total lung capacity followed by exhalation and relaxed breathing just prior to the stimulus to move. On re-analysis, the length of the zone of apposition was measured to $\pm$ 1 mm in sequential frames starting at least five frames prior to the initiation of movement (switch activation) to ten frames after (350 ms). Results were expressed as a change in length from the resting end-expiratory length just prior to the stimulus to move and were based on the mean changes for repeated manoeuvres in each subject.

Recording and data analysis

Electromyographic activity, respiratory pressures, movement of the chest wall and arm movement were sampled at 5 kHz and stored on computer for subsequent analysis. The following bandwidths were used for EMG from the various muscles: costal diaphragm, 16–3200 Hz; transversus abdominis, 53–3200 Hz; crural diaphragm, deltoid and other limb muscles, 16–1000 Hz. The time of onset of EMG activity was identified visually for each EMG trace as the earliest rise in EMG above the baseline level using interactive software that allowed display of each trace individually at high resolution. All onset latencies were measured relative to the stimulus to move. The onset of changes in pressure were identified in a similar way. To determine whether the onset of diaphragm EMG was influenced by the respiratory cycle, the onset of deltoid EMG was related to the phase of respiration which was identified from movement of the upper ribcage (see Fig. 2*A* and *B*). The latency between the onset of each of the parameters was evaluated for individual trials and then averaged for all trials in each subject.

Statistics

The difference between the EMG onsets of the costal and crural portions of the diaphragm, transversus abdominus and deltoid were compared using Student's two-tailed *t* tests. Results are expressed as the mean $\pm$ S.E.M. for the group of subjects, with this mean derived from repeated trials in each subject. Statistical significance was set at the 5% level.

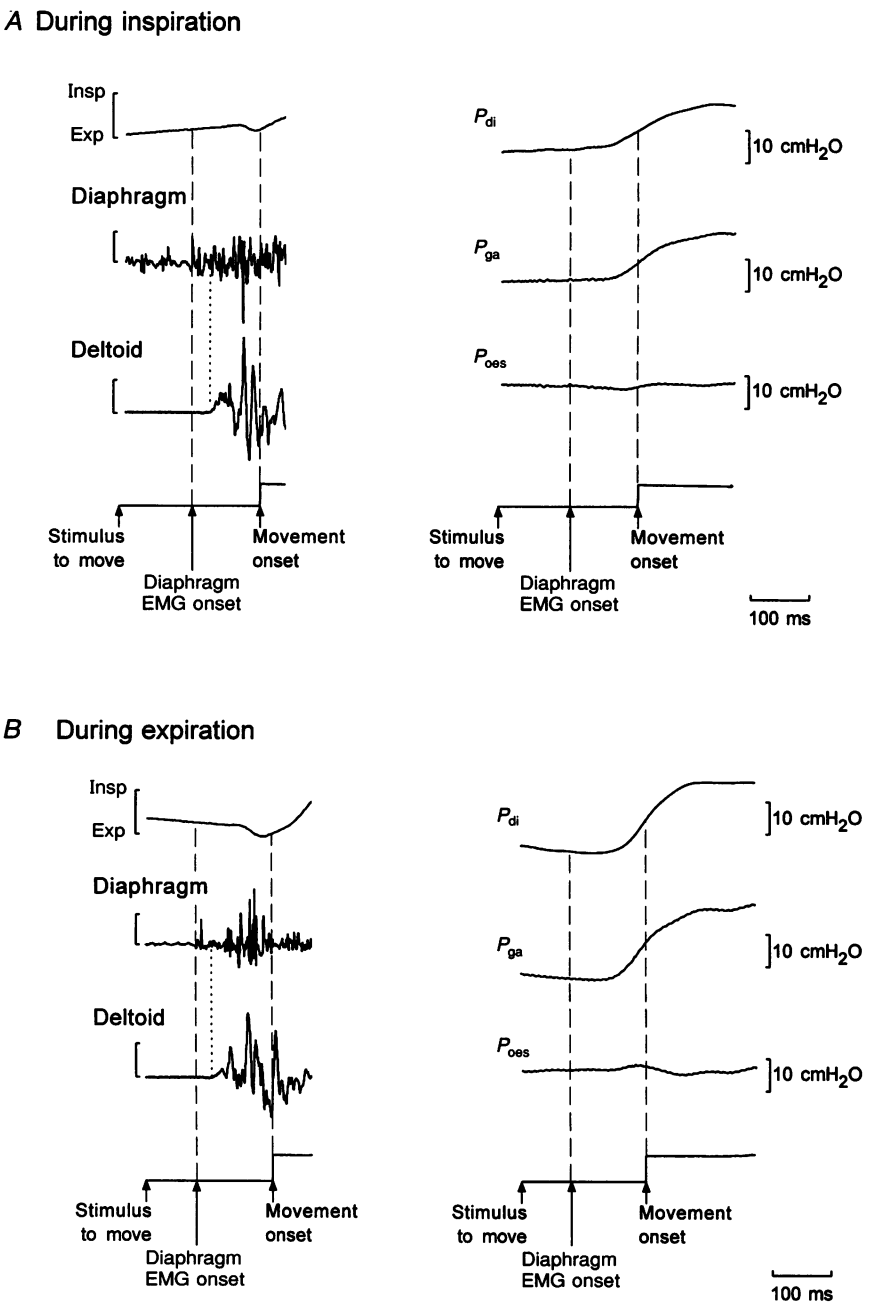

Figure 2. Arm movement during inspiration and expiration

Representative single trials of EMG from one subject from the costal diaphragm and deltoid, transdiaphragmatic (P_{di}), gastric (P_{ga}) and oesophageal pressure (P_{oes}) with rapid shoulder flexion occurring during inspiration (*A*) and expiration (*B*) (indicated by ribcage movement traces (top left panels *A* and *B*)). Onset of diaphragm EMG and initiation of movement of the limb are denoted by the vertical dashed lines and the onset of the increase in deltoid EMG is denoted by the vertical dotted lines. Note the onset of increase in costal diaphragm EMG prior to that of deltoid, the onset of increase in P_{di} and P_{ga} prior to the initiation of movement of the limb and the consistent period between the onset of the increase in costal diaphragm EMG and the onset of deltoid for both inspiration (*A*) and expiration (*B*). Vertical calibrations: diaphragm, 50 μV (*A*) and 100 μV (*B*); and deltoid 1 μV (*A* and *B*). Same time scale for each panel.

RESULTS

Electromyographic responses and arm flexion

When rapid flexion of the shoulder was performed voluntarily in response to a visual stimulus to move, EMG activity in the costal diaphragm increased before any activity was observed in deltoid (by 18 ± 3 ms, mean ± S.E.M.). Data from typical trials in one subject are shown in Fig. 2 for arm movements initiated either during inspiration (*A*) or expiration (*B*). The onset of the increase in diaphragm EMG prior to that in deltoid suggests that this response is preprogrammed by the central nervous system. The latency between the onset of costal diaphragm EMG and the onset of deltoid EMG was not influenced by the phase of respiration (Fig. 2*A* and *B*). Data for each subject (a mean of 10 repetitions) are presented in Fig. 3 for both EMG and mechanical responses. For four of five subjects the increase in EMG of the costal diaphragm occurred significantly before that of deltoid ($t = 4{\cdot}55$, $P < 0{\cdot}002$).

The onset of costal diaphragm EMG preceded the onset of movement of the arm (detected by activation of a switch) by 112 ± 8 ms. The electromechanical delay between the onset of deltoid EMG and the initiation of movement of the arm was 93 ± 6 ms and is consistent with previous reports for this muscle in a similar task (Lee, 1980; Friedli, Hallett & Simon, 1984). The mean latencies between the visual stimulus and the onset of arm movement, the increase in deltoid EMG and the increase in costal diaphragm EMG were 229 ± 6 ms, 140 ± 6 ms and 122 ± 7 ms, respectively. The onset of EMG of the crural diaphragm, recorded using an oesophageal electrode, also preceded that of deltoid (by 17 ± 3 ms), and was concurrent with the onset of activity in the costal diaphragm. The mean difference between the EMG onsets of the two parts of the diaphragm was not significant (7 ± 2 ms; range −20 to +16 ms; $t = 0{\cdot}99$; $P = 0{\cdot}35$).

The time of onset of transversus abdominis EMG preceded that of deltoid by 19 ± 3 ms. Thus, the onset of transversus abdominis and diaphragm EMG occurred almost

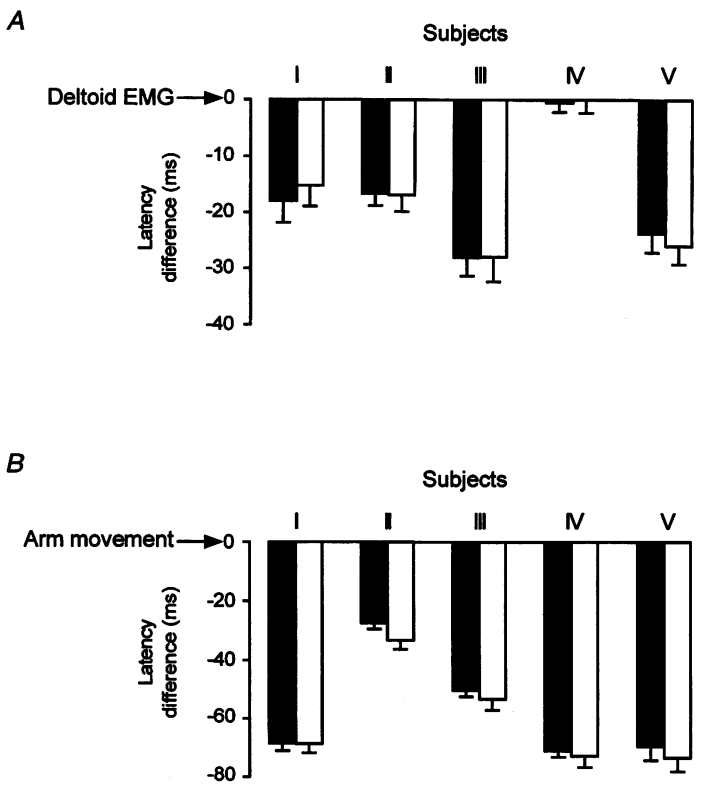

Figure 3. Mean data from all subjects during arm movement

Latency difference between the onset of increase in EMG in costal (■) and crural diaphragm (□) and the onset of EMG in deltoid with the bars aligned to the relative onset of latency of deltoid EMG (*A*) and the onset of increases in P_{dl} (□) and P_{ga} (■) and the onset of arm movement with the bars aligned to the relative latency of movement onset (*B*). Note the concurrent onset of the costal and crural diaphragm EMG, the onset of diaphragm EMG preceding that of deltoid in all subjects except subject IV, and the onset of increases in P_{dl} and P_{ga} prior to the initiation of limb movement.

simultaneously although there was some variability between trials and between subjects. Overall, for the five subjects, the mean difference between the onset of activity in the diaphragm and transversus abdominus was 5 ± 2 ms, range -17 to $+17$ ms; $t = 0{\cdot}56$; $P = 0{\cdot}61$.

Diaphragm activation in response to contraction of distal muscles

Movements of the elbow, hand and thumb were performed to determine whether anticipatory contraction of the diaphragm occurred with all movements of the upper limb or only those associated with large reactive forces. When subjects were standing (as in the main study), rapid movement of either the thumb or wrist was not associated with any anticipatory EMG in the diaphragm (Fig. 4). However, with flexion of the elbow the diaphragm contracted at the same time as for flexion of the arm at the shoulder (see above). This suggests that the early diaphragmatic response when the whole arm moved was more likely to represent a postural adjustment, rather than simply a response associated with the request to 'move as rapidly as possible'.

Respiratory pressures

The initiation of movement of the arm was consistently preceded in repeated trials in all subjects by an increase in gastric and transdiaphragmatic pressures consistent with diaphragmatic contraction. This increase began before the onset of arm movement (Fig. 2) by a mean of 63 ± 7 ms. The delay between the onset of costal diaphragm EMG and the change in transdiaphragmatic pressure was 49 ± 4 ms which is consistent with previous reports based on voluntary movements (e.g. Grillner *et al.* 1978; McGill & Sharratt, 1990). The peak increases of transdiaphragmatic, abdominal and oesophageal pressures were $13{\cdot}8 \pm 1{\cdot}9$, $13{\cdot}5 \pm 1{\cdot}8$ and $0{\cdot}3 \pm 1{\cdot}8$ cmH_2O, respectively. The values for oesophageal pressure were not significantly different from zero. Therefore, the peak transdiaphragmatic pressure was generated purely by the increase in abdominal pressure. The peak transdiaphragmatic pressure occurred 120 ± 7 ms after the initiation of movement of the arm. At the onset of limb movement, the transdiaphragmatic pressure had already reached $25 \pm 3\%$ of its peak value ($\sim 3{\cdot}5$ cmH_2O above the initial resting abdominal pressure).

Diaphragm motion assessed with ultrasonography

In separate studies ultrasonographic images of the length of the zone of apposition of the costal diaphragm were measured before and during rapid flexion of the shoulder in four of the subjects. The absolute length of the zone of apposition (L_{Zapp}) at resting functional residual capacity (FRC) just prior to arm movement ranged from 50 to 100 mm between subjects and different trials (mean, 70 mm). Changes in L_{Zapp} thereafter were expressed relative to the initial length and expressed as a percentage. Based on means from several trials in individual subjects shortening began approximately 30 ms prior to the initiation of movement of the limb (Fig. 5). Due to the relatively slow sampling rate of the ultrasound images (28 Hz) the exact time of onset of this change is imprecise for single trials. The degree of shortening of the diaphragm peaked within ~100 ms of the initiation of limb movement for all subjects (mean, 71 ms) and the diaphragm then lengthened during

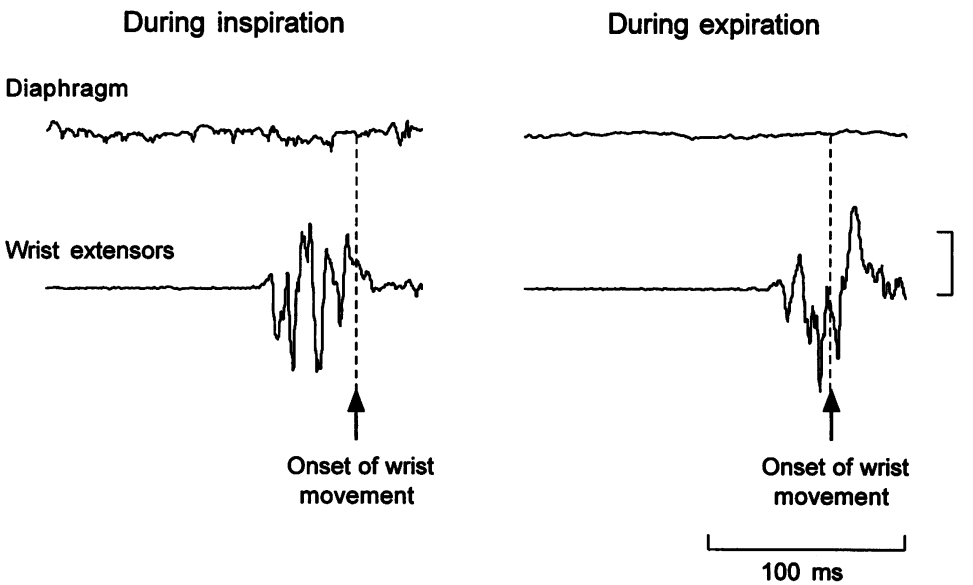

Figure 4. Responses to rapid wrist flexion

Representative single EMG trials of rapid wrist (and finger) extension in response to a visual stimulus to move during inspiration (left panel) and expiration (right panel). Note the absence of change in EMG of the diaphragm in response to the wrist movement. Vertical calibrations: wrist extensors and diaphragm, 300 mV.

the remainder of the arm movement. On average the initial reduction in L_{Zapp} was 8% of initial length and then L_{Zapp} increased to a mean of 12% beyond the initial length at approximately 180 ms after the onset of arm movement. Abdominal (and also transdiaphragmatic) pressure increased progressively during this shortening and lengthening of the diaphragm (Fig. 5). At the onset of diaphragmatic activity abdominal pressure was low enabling diaphragmatic shortening. However, as abdominal pressure increased due to co-contraction of transversus abdominis, further contraction of the diaphragm occurred eccentrically.

DISCUSSION

The results of this study indicate that contraction of the diaphragm contributes to increased intra-abdominal pressure prior to the initiation of movement of large (but not small) segments of the upper limb. The contraction is independent of the phase of respiration. This provides the first direct evidence that the diaphragm may contribute to the postural control of the human trunk in addition to its role in respiration. Furthermore, the findings show that this preparatory contraction of the diaphragm is associated with initial shortening of its muscle fibres and occurs simultaneously with activation of transversus abdominis.

Methodological issues

The monopolar needle electrode used in this study was selective. This was necessary to minimize cross-talk from adjacent muscles (e.g. internal intercostals) but may have resulted in a failure to detect EMG from the motor units with the earliest onset. However, the electrode was commonly repositioned between trials and this increased the number of motor units sampled during the procedure. Any inaccuracy resulting from this would underestimate the difference between the increase in diaphragm EMG and that of deltoid and thus could not alter the main conclusion of the study.

The ultrasonographic evaluation of the length of the diaphragm is also worthy of consideration. Here ultrasonography was applied using a method which gives quantitatively similar estimates of diaphragm length changes as does cineradiography during maximal inspiratory and expulsive efforts (McKenzie *et al.* 1994). In previous studies shortening of the diaphragm was directly measured during maximal isovolumetric contractions of the inspiratory muscles (Gandevia, Gorman, McKenzie & Southon, 1992; McKenzie *et al.* 1994). In the present study the size of length changes accompanying arm movements were well above the limit of resolution and the comparative velocity of the length changes was low. Finally, the use of this technique was designed to show that the diaphragm

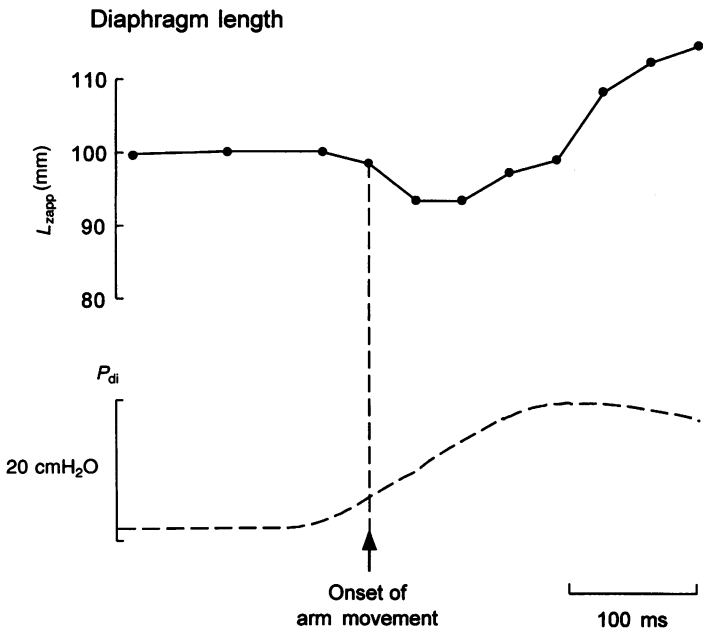

Figure 5. Changes in diaphragm length during arm movement in one subject

Diaphragm length indicated by the length of zone of apposition of the right hemidiaphragm L_{zapp} with ultrasonography (upper panel). Data from the mean of three typical trials in one subject during rapid shoulder flexion. Changes in transdiaphragmatic pressure (lower panel; P_{di}) are taken from a separate study in the same subject (dashed line). The onset of movement of the limb is indicated by the vertical dashed line. The diaphragm begins to shorten before the onset of movement of the limb and it subsequently lengthens. Points indicate measurements of L_{zapp} on alternate frames prior to onset of movement and on each frame after movement onset.

shortened and then lengthened during the rise in intra-abdominal pressure: the absolute magnitude of these changes is not critical.

Diaphragm and posture

Several findings provide support for the proposed relationship between diaphragm EMG and postural control of trunk stability. Firstly, the onset of the increase in diaphragm EMG preceded that of deltoid with a latency appropriate for a preparatory processes associated with limb movement. When a limb is moved reactive forces are imposed on the trunk acting equal and opposite to those producing the movement (Bouisset & Zattara, 1987). The diaphragm cannot move the trunk directly to oppose these forces, but it has been proposed that its contraction contributes to trunk stability via an increase in pressure in the abdominal cavity. Previous studies provide support for this role of elevated gastric pressure (McGill & Norman, 1987; Tesh, Shaw Dunn & Evans, 1987; Cresswell *et al.* 1994). Furthermore, diaphragmatic contraction could increase stability of the trunk by minimizing displacement of abdominal contents into the thorax, thus maintaining the hoop-like geometry of the abdominal muscles. These muscles could then increase spinal stability via tension in the thoraco-lumbar fascia (Farfan, 1973; McGill & Norman, 1987; Tesh *et al.* 1987). Secondly, the failure of contraction of the diaphragm to occur in association with movement of the wrist, fingers or thumb (while it did occur with shoulder and elbow movement) indicates that the diaphragmatic response requires a threshold magnitude of reactive forces resulting from the movement.

Two previous human studies have provided indirect evidence of a contribution of the diaphragm to postural control. These studies documented contraction of the diaphragm prior to contraction of rectus abdominis in preparation for rising onto the toes (Skladal, Skarvan, Ruth & Mikulenka, 1969) and a close relationship between transdiaphragmatic pressure and intra-abdominal pressure with lifting (Hemborg *et al.* 1985). In contrast, previous studies in decerebrate animals have failed to find evidence of a postural role of the diaphragm during evaluation of the response of the diaphragm to stimulation of the cerebellum (Massion *et al.* 1960) and stimulation of tonic cervico-labyrinthine postural reflexes (Massion, 1976).

The contraction of the diaphragm associated with limb movement cannot be mediated by a spinal or supraspinal reflex response to the arm movement since it precedes the motion of the arm and it is too early to be a response to contraction of other posturally activated muscles such as those in the leg. Thus, the response in the diaphragm must be preprogrammed by the CNS and may be initiated as part of the motor command for movement (Cordo & Nashner, 1982; Horak, Esselman, Anderson & Lynch, 1984; Bouisset & Zattara, 1987). The phrenic motoneurone pool may be influenced by corticospinal pathways that do not involve pontomedullary respiratory centres both in animals (Colle & Massion, 1958; Planche & Bianche, 1972) and humans (Gandevia & Rothwell, 1987). Although the present study does not identify the neural structures involved in postural contraction of the diaphragm, the findings provide evidence that the diaphragm is activated via pathways involved in the control of postural activity associated with limb movement. Furthermore, the co-activation of the diaphragm and transversus abdominis identified here is contrary to the normal antagonistic function of these muscles in respiration and this suggests that neural outputs other than those from 'classical' respiratory centres are likely to be involved.

The increase in EMG of the diaphragm preceded the onset of deltoid EMG in all trials regardless of the phase of respiration. This provides evidence that the postural function of the diaphragm interferes with the respiratory activity of this muscle. Previous studies of respiratory muscles have identified variation in respiratory activity of the intercostal muscles due to postural changes (Rimmer *et al.* 1995) and variation in postural activity of the abdominal muscles due to changes in respiratory activity (Hodges *et al.* 1997). The mechanism of this postural–respiratory interaction may involve spinal (Aminoff & Sears, 1971; Kirkwood, 1995) and supraspinal sites (Monteau & Hilaire, 1991; Dobbins & Feldman, 1994). The ventilatory consequence of the interference with the respiratory activity of the diaphragm was not evaluated in the present investigation, but the effect is likely to be limited due to the brief duration of the arm movements (~300 ms).

Although previous animal studies have proposed that the costal and crural portions of the diaphragm may function independently (De Troyer, Sampson, Sigrist & Macklem, 1981; van Lunteren, Haxhiu, Cherniack & Goldman, 1985), the co-activation of the costal and crural portions of the diaphragm in the present study suggests that both regions of the diaphragm function together for their role in postural control.

The magnitude of increase in abdominal pressure ($13{\cdot}5 \pm 1{\cdot}8$ cmH_2O) in the current study is in the same range as that reported previously during walking (11–30 cmH_2O; Grillner *et al.* 1978), but less than that associated with landing from a jump (40–121 cmH_2O; Grillner *et al.* 1978), lifting (54–81 cmH_2O; Troup, Leskinen, Stålhammer & Kuoriaka, 1983), trunk loading (87–133 cmH_2O; Cresswell *et al.* 1994) and during a maximal forced expiration against a closed glottis (over 100 cmH_2O) (Gandevia *et al.* 1992; Goldish, Quast, Blow & Kuskowski, 1994). However, the magnitude of the perturbation to the trunk resulting from the latter manoeuvres is likely to be greater than that associated with arm movements. The increase in gastric pressure associated with arm movement and the negligible change in oesophageal pressure indicates that the increase in trans-diaphragmatic pressure is predominantly due to the contraction of the diaphragm and abdominal muscles. The diaphragm shortened initially consistent with an increase in

activation but then lengthened as abdominal pressure rose. This suggests that the diaphragm contracts eccentrically following contraction of transversus abdominis and the other muscles acting on the abdomen (e.g. pelvic floor muscles). However, the length changes in either direction were relatively small, approximately 10% of initial diaphragm length.

In summary, the findings of this study show that contraction of the diaphragm precedes the onset of movement of the limb and support the hypothesis that this preparatory action may aid truncal stability.

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